Published in final edited form as:

Nestle Nutr Inst Workshop Ser. 2016 ; 85: 59–69. doi:10.1159/000439487.

Interrupting Intergenerational Cycles of Maternal Obesity

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Abstract

Factors operating in the preconception and prenatal periods, such as maternal obesity, excessive gestational weight gain, and gestational diabetes, predict a substantial fraction of childhood obesity as well as lifelong adverse health consequences in the mother. These periods may lend themselves to successful intervention to reduce such risk factors because parents may be especially willing to change behavior if it confers health advantages to their children. If effective interventions started before or during pregnancy can be maintained after birth, they have the potential to lower the risk of both maternal obesity in the next pregnancy and obesity in the growing child, thus helping to interrupt maternal and child inter-generational vicious cycles of obesity, diabetes, and related cardiometabolic health consequences. While this paradigm is appealing, challenges include determining the magnitude, causality, and modifiability of these risk factors, and quantifying any adverse consequences of intervention.

Women who are obese entering pregnancy, especially those who gain excessive weight during gestation, tend to retain more weight postpartum, putting them at higher risk of later diabetes and cardiovascular disease. If they become pregnant again, they enter pregnancy at a higher weight status, reigniting this cycle. Excessive gestational weight gain and gestational diabetes, both more common among obese women, are associated with obesity in the next generation; childhood obesity portends many adverse health outcomes. Moreover, if the obese child is female, she is likely to enter any future pregnancies obese, thus perpetuating intergenerational cycles of obesity and its consequences (Figure 1). One could consider interrupting the cycles either once pregnancy starts—on moderating excessive weight gain or preventing or treating GDM—or before pregnancy by having women enter pregnancy at a lower weight status. In this article, I review evidence for the existence of these vicious cycles, which may very well be driving increasing rates of obesity and noncommunicable disease around the globe, and for promising ways to interrupt these cycles.

Maternal Obesity

In almost all regions of the world, obesity prevalence has increased among women in the past 30 years.^[1] In the United States, no nationally representative data exist on prepregnancy obesity. However, NHANES data show that rates of overweight (BMI 25–29.9 kg/m²) and

obesity (BMI > 30 kg/m²) among women age 20–39 years, the prime reproductive period, approximately tripled from the 1970s to 2005.^[2] By 2009–2010 almost one-third of women in this age group were obese, and more than half were overweight or obese. Among pregnant women in 20 states participating the Pregnancy Risk and Monitoring System (PRAMS), pre-pregnancy obesity rates rose from 17.6% to 20.4% just from 2003 to 2009.^[3] In the CDC's Pregnancy Nutrition Surveillance System of over 1 million low-income women, pre-pregnancy obesity prevalence rose from 34.6% to 53.7% from 1988 to 2011, mirroring the overall US data for reproductive age women.^[4]

These trends are disturbing because overweight and obesity entering pregnancy are associated with several adverse pregnancy outcomes, including gestational diabetes (GDM), gestational hypertension and preeclampsia, and cesarean section, each of whose rates increased over the past 2–3 decades.^[5–10] GDM is associated with higher lifetime risk of developing type 2 diabetes in the mother, and preeclampsia is associated with later cardiovascular disease.^[11, 12] Type 2 diabetes and cardiovascular disease are leading causes of death in the developed world and have emerged in the developing world as well.^[13, 14] These “noncommunicable diseases” now represent the largest fraction of deaths in all regions of the world except the lowest income. By 2030 approximately half a billion people will have type 2 diabetes.

Obesity in the mother is also strongly related to obesity in the child. Childhood obesity not only presages adult obesity, diabetes and cardiovascular disease, but also gives rise to adverse consequences in the child.^[15, 16] These include psychosocial sequelae, asthma, musculoskeletal problems, and even type 2 diabetes among a small proportion of adolescents.

Gestational weight gain (GWG)

In 2009, the US Institute of Medicine promulgated new recommendations for weight gain during pregnancy, which for the first time included guidelines for obese women (Table 1). They recommend that lighter women gain more than heavier women. Gain below each recommended range is called inadequate, within the range adequate, and above the range excessive. In the CDC's Pregnancy Nutrition Surveillance System, rates of excessive GWG rose from 37.4% in 1988 to 48.0% in 2011, and inadequate gain dropped from 33.0% to 21.0%.^[4] Overweight and obese women are more likely than their leaner counterparts to gain excessively.^[17]

Excessive GWG is associated with childhood obesity. One of the first studies, from the US pre-birth cohort study Project Viva, showed that 3-year-old children of mothers who gained excessively had a mean BMI z-score of 0.52, compared with 0.17 for inadequate gainers.^[18] This study was followed by an analysis from the Growing Up Today Study, showing that once adjusted for maternal BMI, higher amounts of GWG were linearly associated with BMI in the offspring at the age of 9–14 years (Figure 2).^[19] A recent meta-analysis estimated that excessive weight gain during pregnancy (defined somewhat differently across the 12 included studies) was associated with an odds ratio of 1.3 (95% CI 1.2, 1.5) for obesity in children ranging in age from 2 to 18 years.^[20]

While these observational studies confirm strong associations, they cannot confirm whether excessive GWG actually causes offspring obesity. One of the best ways to judge causality, magnitude of effect, and ability to change behavior in the real world is via randomized controlled trials (RCTs) of behavior change to modify GWG, with child followup. In general, interventions to change lifestyle behaviors during pregnancy offer some advantages. Women may be especially willing to change behavior for the health of the growing fetus. Pregnancy is a few months long; weight control in non-pregnant adults is very often successful for the first few months. Given that the prenatal period is one of frequent clinical visits, one can imagine delivery system changes to enhance intervention effects. In addition, interventions started during pregnancy could be continued after birth, for the benefit of both mother and child. Nevertheless, we must be careful not to pin blame on mothers if they are unable to change behaviors in our ubiquitous obesogenic environment.^[21] It is important to consider interventions that at least take the social context of the mother into account if not change the environment itself.

Unfortunately, for the case of GWG and offspring obesity, the RCT evidence base is slim to date. Before 2014, only 10 RCTs, comprising just over 1000 total participants, had attempted to reduce GWG among overweight or obese pregnant women.^[22] The pooled effect of these trials was a reduction of 2.21 (95% CI -2.86, -1.57) kg, but none followed the offspring. 2014 saw the publication of two such RCTs that are funded to follow the children to at least 1 year of age, but to date have reported results only until shortly after delivery. These two studies, LIMIT in Australia and Healthy Moms in Oregon (US), had many features in common (Table 2).^[23, 24] Among the goals of both was to achieve GWG that is actually lower than the Institute of Medicine recommends. The major differences were that LIMIT involved individual intervention, whereas in Healthy Moms the primary intervention modality was weekly group visits; and that LIMIT had a much larger sample size. Perhaps surprisingly, Healthy Moms demonstrated reductions in GWG and large-for-gestational age births whereas LIMIT did not affect either. LIMIT did show a modest reduction in macrosomia (Tables 3,4). We await results from childhood followup from both of these studies as well as other RCTs that are ongoing.

Gestational diabetes (GDM)

In rodent experiments, inducing GDM causes macrosomia in the offspring, followed by adiposity in the growing animal. If the animal is female, when she gets pregnant she is more likely to get GDM, causing obesity and GDM in the next generation, *ad infinitum*.^[25] Given their longer lifespan and genetic and environmental variability, proving that such cycles exist in humans is more difficult. Two sib-pair studies suggest a causal relationship between diabetes during pregnancy (either pre-existing diabetes or GDM) and higher BMI in the offspring, at least from school age to young adulthood. One of them consisted of 58 sibs from 19 Pima (American) Indian families, a population with endemic obesity and diabetes.^[26] The other, albeit comprising 46,000 brothers, was limited to male Swedish conscripts whose obesity rates were low and whose mothers had low rates of obesity and diabetes.^[27] The few observational studies in which the investigators adjusted for maternal BMI also do not show an association. In a 2011 meta-analysis of three studies, the pooled estimate was only 0.07 (-0.15, 0.28) higher BMI z-score among offspring of women with

gestational diabetes v. no diabetes.^[28] Another study that adjusted for maternal BMI, an analysis of Growing Up Today Study cohort comparing mothers with GDM v. no diabetes, showed an odds ratio of only 1.2 (95% CI 0.8, 1.7) for obesity among 14,000 9–14-year-olds.^[29] Because the mothers are all registered nurses who might be especially attuned to adequate GDM treatment, this modest result raised the possibility that treatment of GDM treatment in pregnancy moderates the otherwise higher risk of obesity in the offspring.

The Australian Carbohydrate Intolerance Study in Pregnant Women (ACHOIS), an RCT of treatment of mild-to-moderate GDM, resulted in reduction of macrosomia and large-for-gestational age, as did another large trial in the US.^[30, 31] The hope was that reduction in large babies would translate into reduction in large children. In a clever merging of the ACHOIS trial data with data from a South Australian statewide surveillance system of heights and weights among 4–5 year-old children, however, BMI was not lower among children of mothers in the intervention v. control groups.^[32] Several explanations for this null result could exist. One of the more intriguing is that GDM causes fatter newborns, but this excess fat tends to disappear during infancy.^[33] Some data suggest that the GDM-offspring obesity connection only reappears during school age and later,^[34, 35] suggesting that a “second hit” of some sort is needed to manifest this phenomenon.

In addition to treatment of GDM, attention is also focused on its prevention. Some ongoing RCTs aim to modify the diet of pregnant women irrespective of its impact on weight gain.^[36] Nevertheless, interventions once pregnancy starts may be a drop in the bucket of GDM prevention. In the Project Viva cohort, in which the investigators measured diet during the first trimester with a validated food frequency questionnaire augmented by interviews for vitamins and supplements, on the whole neither nutrients nor foods nor food groups nor dietary patterns nor even gestational weight gain predicted GDM.^[37, 38] By far the largest predictor was higher pre-pregnancy BMI. Other studies suggest that GWG is not a prime determinant of GDM,^[39] and universally cohort studies confirm that pre-pregnancy BMI is a strong predictor.^[40]

In summary, interventions to curb excessive GWG and to prevent (or treat) GDM are promising strategies to lower the risk of maternal and childhood obesity and thus interrupt the intergenerational cycles, but they are not yet proven.

Pre-pregnancy obesity

As reviewed above, entering pregnancy at an ideal (v. higher) body weight is associated with better pregnancy and childhood outcomes. Thus, it might seem to “just make sense” to treat obesity in women who will soon get pregnant. However, it is not clear that maternal obesity causes childhood obesity via its programming effects during pregnancy, rather than through genetic inheritance or shared post-natal environment. The Swedish sib-pair study suggests not,^[27] as does a previous paper using the FTO gene as an instrumental variable in Mendelian randomization.^[41]

In addition, treating pre-conception obesity could carry risks. Rapid short-term weight loss in an adult can have adverse effects;^[42, 43] effects on the conceptus are unknown. In the US,

it is difficult to recruit women in the pre-conception period as half of pregnancies are unplanned. Unplanned pregnancies are more common among minorities and women of lower socioeconomic status, who both have more preconception risks including obesogenic risk factors and are less likely to agree to be involved in research studies.^[44–47] Although it is attractive to address preconception issues in the interpregnancy period, such studies miss the large proportion of women who have not yet had their first pregnancies. To date preconception cohort studies are rare, and no intervention studies of weight loss exist.^[48] A need exists to study preconception determinants—from society to biology—of outcomes in early and late pregnancy and of longer-term maternal and offspring outcomes. Given recent animal experimental studies showing paternal effects on pregnancy outcomes,^[49] preconception studies would do well to include biological as well as social influences of fathers. A range of intervention studies is needed, to address benefits and risks in highly controlled studies (efficacy studies), how to achieve effectiveness in real-world settings, and once proven effective, how to scale them for maximum impact.

Conclusion

Interventions starting in pregnancy, and perhaps before, have the potential to reduce risk factors like pre-pregnancy obesity, excessive GWG, and GDM, and thus interrupt intergenerational vicious cycles of maternal and child obesity. However, the implications for clinical and public health practice require additional evidence from observational and intervention studies that follow children and mothers after delivery.

Acknowledgments

Funded in part by a grant from the US National Institutes of Health (R37 HD 034568)

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Figure 1.
Inter-generational vicious cycles of maternal obesity

Figure 2.

Association of gestational weight gain with offspring body mass index, before and after adjusting for maternal BMI. Data from the Growing Up Today Study.¹⁹

Table 1

Institute of Medicine 2009 Gestational Weight Gain Recommendations

BMI Category	BMI (kg/m ²)	Recommended total gain	
		Lb	Kg
Low	<18.5	28–40	12.5–18
Normal	18.5–24.9	25–35	11.5–16
Overweight	25–29.9	15–25	7–11.5
Obese	30+	11–20	5–9

Table 2

Two randomized trials of behavior change to limit gestational weight gain, LIMIT²³ and Healthy Moms,²⁴ had many similar features.

Feature	LIMIT	Healthy Moms
Enrolment	10–20 weeks	< 20 weeks
BMI eligibility	> 25 kg/m ²	> 30 kg/m ²
Weight gain goal	0–5 kg	+/- 3%
Intervention led by dietitian	Yes, then research assistant	Yes, then group leader
Diet: theory-based, tailored, includes calorie calculation	Yes Low glycemic index	Yes DASH
Clinician involvement	Passive	Passive
Individual v. group	Individual	Group
Sample size	2200	120

Table 3Selected Results from LIMIT²³

	Lifestyle advice N=1080	Standard care N=1073	Adjusted treatment effect
	<i>Mean (SD)</i>		<i>β (95% CI)</i>
Total GWG, kg (36 wk gestation)	9.39 (5.74)	9.44 (5.77)	−0.04 (−0.55 to 0.48)
	<i>N (%)</i>		<i>RR (95% CI)</i>
Excessive GWG	380/897 (42)	368/871 (42)	0.99 (0.89 to 1.10)
Preterm birth	62 (6)	83 (8)	0.74 (0.54 to 1.02)
C-section	370 (34)	389 (37)	0.95 (0.85 to 1.06)
Preeclampsia	56 (5)	53 (5)	1.03 (0.71 to 1.47)
GDM	148 (14)	120 (11)	1.21 (0.96 to 1.52)
LGA	203 (19)	224 (21)	0.90 (0.77 to 1.07)
Macrosomia >4 kg	164 (15)	201 (19)	0.82 (0.68 to 0.99)

GWG, gestational weight gain

GDM, gestational diabetes

LGA, large-for-gestational age

Table 4Selected Results from Healthy Moms²⁴

	Intervention N=56	Control N=58	Adjusted treatment effect
	<i>Mean (SD)</i>		β (95% CI)
Total GWG, kg (3 wk postpartum)	1.2 (5.6)	-2.6 (5.5)	-3.8 (-5.9 to -1.7)
	<i>N (%)</i>		<i>RR (95% CI)</i>
Excessive GWG/wk	24 (44%)	47 (82%)	[Not reported]
Preterm birth	4 (7%)	1 (2%)	4.4 (0.4 to 220)
C-section	21 (38%)	26 (45%)	0.74 (0.35 to 1.56)
Preeclampsia/GH	5 (9%)	6 (10%)	0.85 (0.24 to 2.96)
GDM	6 (11%)	7 (12%)	0.87 (0.28 to 2.78)
LGA	5 (9%)	15 (26%)	0.28 (0.09 to 0.84)
Macrosomia >4 kg	6 (11%)	13 (22%)	0.42 (0.15 to 1.18)
SGA	3 (5%)	4 (7%)	0.76 (0.11 to 4.76)

GWG, gestational weight gain

GDM, gestational diabetes

LGA, large-for-gestational age

SGA, small-for-gestational age